after the beginning of the response of the quadriceps and the gastrocnemius respectively.

This contraction of the antagonists forms part of the spinal reflex pattern elicited by the tendon stimulation, and such a reflex pattern is made use of in voluntary movements of a like nature to the reflex movement.

In conclusion we wish to express our thanks to Sir Frederick Mott for the facilities that he afforded us for carrying out these experiments in his laboratory at the Maudsley Hospital. We are, in addition, deeply indebted to him for much valuable advice.

An Abnormal Relationship of the Electrical to the Mechanical Response in the Ventricles.

By J. Crighton Bramwell, M.B., M.R.C.P.

(Communicated by Prof. A. V. Hill, F.R.S. Received June 1, 1922.)

(From the Royal Infirmary, Manchester; working for the Medical Research Council.)

[Plate 1.]

The record described in this paper was made on a man aged 64, who was suffering from complete heart block (Plate 1, fig. 1). The characteristic features peculiar to it were observed on one occasion only, although numerous other records were made on the same subject. It was impossible to continue the observations as the patient developed acute tonsillitis and died of œdema of the glottis.

The observation in question is shown in Plate 1, fig. 2, in which the lower record is an electrocardiogram taken with a sensitive string at about one-eighth of the usual magnification, and the upper record is a combined carotid and radial sphygmogram taken by a method previously described by Bramwell and Hill (5), employing a hot-wire sphygmograph (1) (2). In this particular instance the carotid receiver was placed low down in the neck, and consequently not only arterial but also certain additional waves from the internal jugular vein were recorded.

The spacing of the ventricular complexes of the three electro-cardiograms differs from that of the three arteriograms. For purposes of comparison it is convenient to select similar points in the three cycles; and those which present the greatest facility for accurate measurement, in this record, are the commencement of S in the electrocardiogram, and the commencement of the radial upstroke in the hot-wire sphygmogram. Measuring* from these two points in each of the three cycles it is found that the intervals between the ventricular complexes are 1·828 sec. and 1·810 sec. respectively; whereas the intervals between the arteriograms are 1·521 and 2·135 sec. This discrepancy is due to the fact that the interval between the commencement of S in the electrocardiogram and the radial upstroke in the hot-wire record (which for convenience will be referred to as the "S-Radial interval") is 0·268 sec. in the case of the second cycle, whereas in the first and third cycles it is 0·575 and 0·593 sec. respectively. Comparing these values of the S-Radial interval with those obtained from other records taken from the same patient (of which fig. 3, Plate 1, is a typical example), it appears that even the interval in the second cycle is unduly prolonged, whereas those in the first and third cycles are of such duration as to fall far beyond the limits of normal variation. In the three cycles in Plate 1, fig. 3, the S-Radial intervals are 0·152, 0·148, and 0·160 sec. respectively.

Discussion.—The S-Radial interval may be sub-divided into three parts; first, the interval between the commencement of the electrical response, and that of the mechanical response in the ventricle; secondly, the period of isometric contraction of the ventricle prior to the opening of the aortic valve; and, thirdly, the time occupied by the conduction of the pulse wave from the aortic orifice to the radial artery. The normal velocity of the pulse wave in the upper limb of this patient, as calculated from other records, varies from 6·9 to 7·2 metres per second, and it is impossible to believe that more than a very small part of the long time difference, which in the record in question amounts to 0·4 sec., can be accounted for by increased delay in the transmission of the pulse wave. One is forced therefore to conclude that the increased duration of the S-Radial interval in the cycles referred to, must have been due to an increase in the time which elapsed between the excitation wave accompanying the electric change and the opening of the aortic valves. Either, therefore, the latent period was exceedingly long (0·4 sec. longer than usual) or the development of the isometric response was very slow.

It has been shown by Mines (3) that under certain experimental conditions in the frog's heart an electrical response may be obtained without any subsequent mechanical response, and that under similar conditions, before the mechanical response is completely abolished, there appears an increase in the interval between the contraction of the auricle and that of the ventricle

---

* All measurements referred to in this paper were made with the Lucas comparator (6) (7).

This increase is partly accounted for by a prolongation of the time which elapses between the excitation of the ventricle and its mechanical response. It has also been shown by Weizsäcker (4) that an excitation may be followed by a thermal response without any subsequent mechanical response. There appears however to be no record in the literature of a similar prolongation of the interval between the electrical and the mechanical responses in the human subject.

Making due allowance for the coincidence of P with R and S in the second cycle, the initial deflections of the three ventricular complexes in this record are exactly similar to one another, and exhibit no deviation from those seen in other records taken from the same patient. Further, the isoelectric phase of the ventricular complex is not disturbed by any adventitious deflections such as would be present if the normal rhythm were interrupted by the presence of an extra-systole. There is no ground, therefore, for supposing that the irregularity exhibited by the arterial pulse can be explained by the incidence of supplementary impulses, and there can be no doubt that each of the three arteriograms belongs to the same cycle as the rapid initial deflection of the ventricular complex in the corresponding electrocardiogram.

The record of fig. 2, Plate 1, shows certain features which suggest either (*a*) that the right ventricle has responded in the normal fashion, and that the abnormal time relations exhibited are due to the left ventricle; or (*b*), that a very slow development of the later stages of the mechanical response has caused the abnormal delay.

On the hot-wire record there appear three small peaks (×, ×, ×) which exhibit a striking similarity to one another, and bear a very definite relationship to the commencement of the ventricular complex, a relationship which suggests that they must be associated with ventricular systole. The time interval which elapses between the commencement of S in the electrocardiogram and the tip of these peaks is 0·089, 0·093, and 0·089 sec., in the three cycles. This interval is slightly longer than that which elapses between the commencement of S and the carotid upstroke, which in other records varies from 0·068 to 0·078 sec. It is distinctly shorter than the average time interval (0·18 sec.) which elapses between the summit of P in the electrocardiogram and the "*a*" peak in the four isolated auricular cycles of fig. 2. Further, there is a striking resemblance between these peaks and the "*a*" waves in the hot-wire record. These peaks may be due to venous waves associated with systole of the right ventricle, waves which are produced either by regurgitation from ventricle to auricle during the process of closure of the A—V valve, or by bulging of that valve into the auricle. If this hypothesis be correct, these waves would start in the auricle at the very commencement of ventricular systole; that is to say, considerably earlier than the arterial waves due to systole of the left ventricle, which do not reach the aorta until the end of the isometric period of ventricular contraction. The velocity of transmission of these waves, however, would be relatively low, owing to the low pressure pertaining in the venous system, and the adverse direction of the blood stream. Taking these facts into consideration, namely, the early phase in the cardiac cycle at which such a wave would start from the auricle, and the relatively low velocity with which it would be transmitted along the veins, the probable time of arrival of a wave of this nature in the neck is not inconsistent with the time relations exhibited by these three peaks in the hot-wire record.

If, therefore, this interpretation of the record be correct, one is forced to accept the possibility either of an abnormally slow development of pressure in the left ventricle, or of an abnormally long interval between the mechanical response of the right ventricle and that of the left.

Summary.

1. A record is described, in two cycles of which the time interval between the ventricular complex of the electrocardiogram and the arrival of the pulse wave in the arterial system is greatly prolonged.

2. The delay appears to be due, either to a marked prolongation of the latent period, or to a very slow development of the mechanical response in the left ventricle.

3. Certain features of the record suggest that the right ventricle has responded in the normal fashion.

My thanks are due to Dr. Charles Melland for the facilities granted me in making these observations, and to Dr. T. Coögan for bringing the case to my notice.

REFERENCES.

(1) Hill, A. V., 'Journ. of Physiol.,' vol. 54, Proc., liii (1920).
(2) Hill, A. V., 'Journ. of Physiol.,' vol. 54, Proc., cxvii (1921).
(3) Mines, G. R., 'Journ. of Physiol.,' vol. 46, p. 230 (1913).
(4) Weizsäcker, J., 'Journ. of Physiol.,' vol. 48, p. 396 (1914).
(5) Bramwell, J. C., and Hill, A. V., 'Lancet,' vol. 202, p. 891 (1922).
(6) Lewis, T., 'Heart,' vol. 7, p. 117 (1920).
(7) Lucas, K., 'Journ. of Physiol.,' vol. 39, p. 217 (1909).

DESCRIPTION OF PLATE 1.

FIG. 1.

Electrocardiogram, lead III.
Time intervals, 0·2 sec.
Record reads from left to right.

FIG. 2.

ECG. = Electrocardiogram, lead III.
H.W. = Hot Wire sphygmogram.
Rad = radial primary deflection.
Car = carotid primary deflection.
a = isolated auricular venous wave.
Dotted line = S-Radial interval.
Time intervals = 0·2 sec.
Record reads from right to left.

FIG. 3.

ECG. = Electrocardiogram, lead III.
H.W. = Hot Wire Radial Sphygmogram.
Time intervals = 0·2 sec.
Record reads from right to left.

Alterations in the Diameter of Erythrocytes during Hæmolysis.

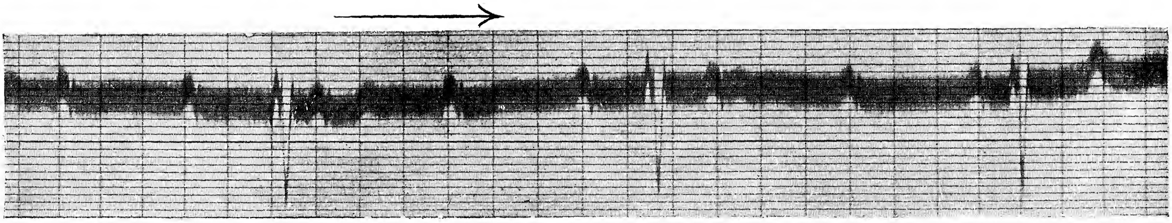
By ERIC PONDER.

(Communicated by Sir E. Sharpey Schafer, F.R.S. Received June 7, 1922.)

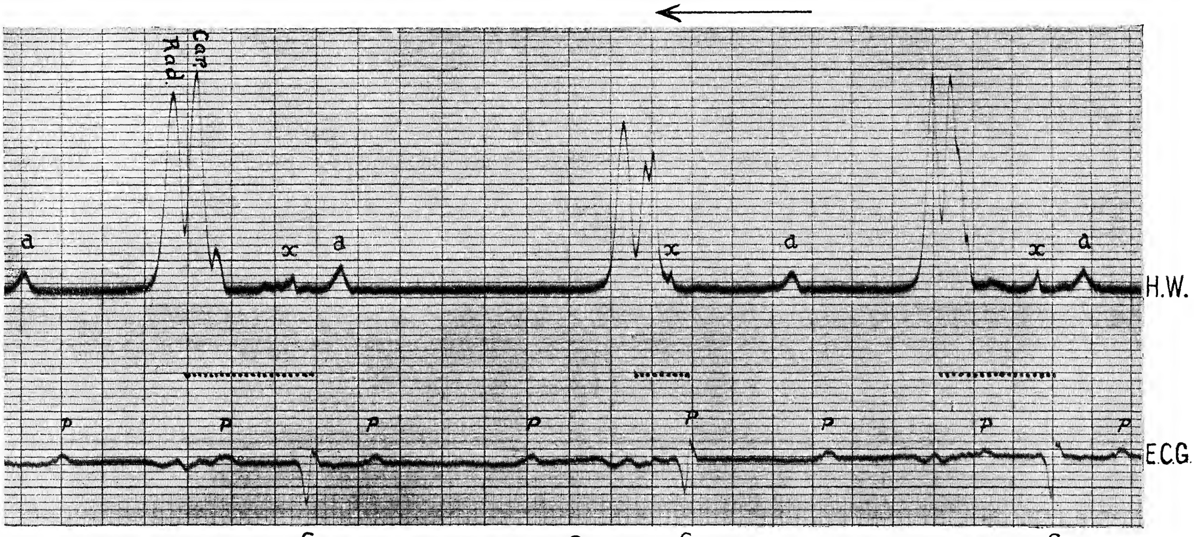
(From the Department of Physiology, Edinburgh University.)

Introduction.

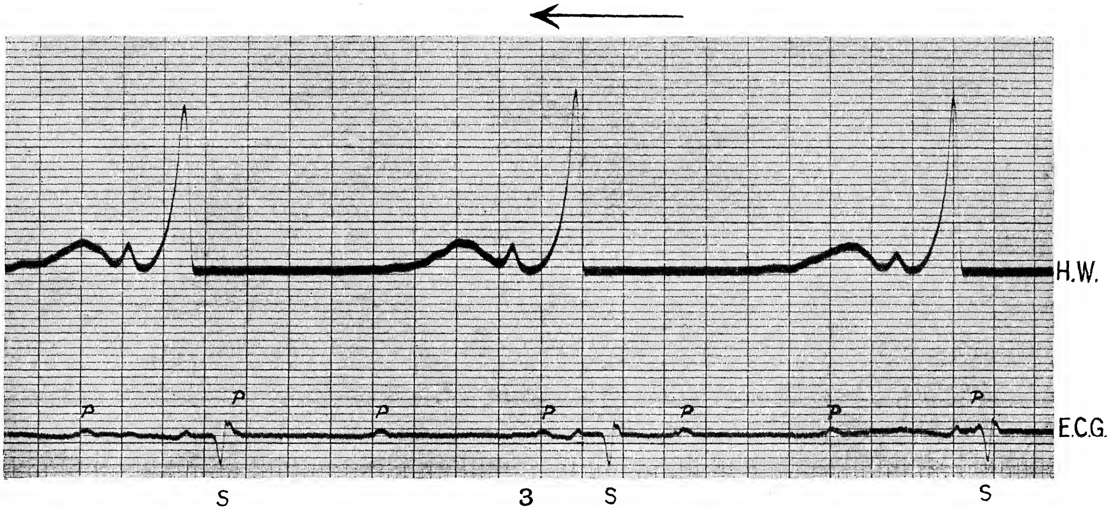
There has been some doubt as to the manner in which certain substances produce hæmolysis, especially saponin, bile salts, and those hæmolysins which are the result of immunity experiments. The doubt has even been extended to the mode of action of hypotonic saline, the hæmolytic action of which is generally put down to the osmotic changes which it produces. Bechhold (1), however, considers that surface tension phenomena have much to do with this hæmolysis. As regards saponin, some observers consider that it destroys the cell wall (Arrhenius (2)), whereas others consider that it produces hæmolysis by lowering of surface tension; others, again, deny that there is any relation between surface tension and hæmolytic activity in the case of the saponins (Woodward and Alsberg (3)). The same doubt prevails regarding bile salts, it being considered by many that they dissolve the lipoid envelope, and by others that surface tension plays an important part. As regards hæmolysis by hæmolytic amboceptors, it is suggested by some that the action is due to a



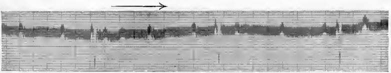
1



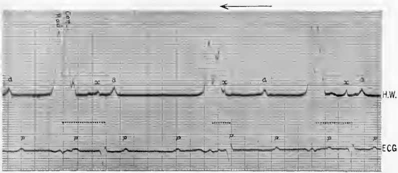
2



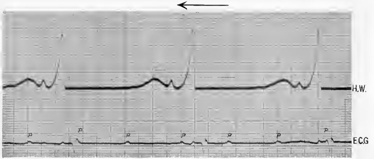
3



1



2



3